The Canadian
Medical Association Journal

Vol. XIV TORONTO, JUNE, 1924 No. 6

An Address

ON

SOME EFFECTS OF TEMPERATURE ON THE BLOOD AND CIRCULATION*

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THE influence of temperature upon the animal organism has been vaguely conceived for many centuries, but it may be said that its use as a therapeutic agent has been based upon empirical observations. Of recent years, however, much work has been done to investigate the effect of heat and cold upon the functions of the animal body. This work has been undertaken, in the main, to elucidate the effect of certain abnormal conditions of environment or to study the manner in which the body responds to external stimuli. In other words, by what means does it adapt itself to changes of temperature. In few of these investigations has the therapeutic effect of heat and cold been under consideration. Nevertheless, the knowledge obtained through these experiments when reviewed with this point in view throws a great deal of new light upon the indications for its use, and the effects likely to be produced by this ancient and valuable therapeutic agent. It was not to be expected that the means employed to produce the temperature changes should be always the same. In consequence the results sometimes appeared conflicting, until it was appreciated that other physical characters of the agent, besides that of temperature, might have an important influence in producing some of the changes observed. On considering the therapeutic influence of temperature it may be well at the outset to review the changes in the general condition of the organism when the internal temperature of the body is altered. Probably one of the most important results of such a change is that upon the general metabolism. Krogh[1] has given an excellent review of the effect of changes of body temperature upon metabolism. In general the experiments would undoubtedly indicate that the general metabolism increases in direct ratio to the increase of body temperature, until a maximum is reached when the metabolism begins to decline. This decline seemed to be due to a deleterious effect which an excessive increase of temperature had upon the vitality of the organism. When the surface of the body is exposed to low temperatures or when the cooling powers of the environment be greatly increased there is a pronounced acceleration of the metabolism. As a result of the cooling the thermogenic power is increased. This may occur at such a rate as to produce a distinct rise in the rectal temperature in spite of the heat loss. If the exposure be too severe and continued the thermogenic power becomes depressed and the body temperature and metabolism begin to fall. This subject has been carefully investigated by Lefèvre, Rubner, Leonard Hill and others.

In man, when an increase of body temperature occurs, as in fever due to infectious processes, there is a definite increase in the meta-

*Read before the Medico-Chirurgical Society, Harrogate, April 12th, 1924.

bolic rate. This increase of metabolism during infective fevers has been shown by Du Bois[6] to be directly proportional to the rise in temperature, being 13 per cent. higher for each degree centigrade above normal. This accords very closely to the temperature law of van't Hoff. In thermal fever produced by immersion in a hot bath, Koehlar[7] has shown a similar increase in oxygen consumption. Whether the increased metabolism and fever are both due to a common cause or are interdependent has not been definitely determined.

Practically all experiments upon normal man, in regard to the influence of changes of body temperature, have been carried out between 37°C. and 41°C. Such elevations of temperature have usually been produced by immersion of the body in hot water or in hot air or electric cabinets. As might be expected the results were modified by the character of the medium by which the increase of body temperature was produced.

Hill and Flack[8] were the first to make a study of the effect of thermal fever upon the human body. They found on immersion up to the neck in hot water that the body temperature could be appreciably increased. With this increase there was tachycardia, increased pulse pressure, hyperpnoea, and fall in carbon dioxide partial pressure in the alveolar air with increase of that of the oxygen. They also found that a healthy individual could hold his breath 2½ times as long as normally. A partial immersion immediately lessened all these effects while a cold shower practically reversed all the phenomena except the rectal temperature which remained elevated. Experiments carried out by Bazett and Haldane[2] are also of much interest. They immersed healthy men up to the neck in a bath in which the water was kept circulating at a constant temperature. They were able to keep the body temperature steady or raise it at will to any desired point or at any rate. At temperatures above 37° C. they found the body temperature to be slightly greater, up to 0.2°C., than that of the water.† At 37°C. the respiration remained normal but the pulse rate slightly increased while both the systolic and diastolic blood pressure fell (10 - 20 mm. Hg.) The most obvious result was a conspicuous diuresis which reached its maximum during the second half hour as a rule, and the normal flow was not re-established for 3 or 4 hours after immersion. The quantitative chemical character of the urine did not materially change although qualitatively it gave all the expected results of dilution. This diuresis was not apparently the result of absorption of water but was probably due to the tissue fluids being squeezed into the circulation by the pressure of the water.

A rapid increase of body temperature produced pronounced respiratory disturbances. These were in proportion to the rate at which the temperature was raised. The first sign was hyperpnoea. In one experiment in which the temperature was raised at a rate of 0.13°C. per minute the expired air increased from 6 litres to 27.3 litres per minute. There was a rapid rise in the respiratory quotient to 1.3, and the alveolar carbon dioxide fell from a partial pressure of 38.7 to 25.6 mm. During this period there developed faintness, mental confusion and tingling. These latter symptoms could be relieved or prevented by inhaling a mixture rich in carbon dioxide or in oxygen. The authors concluded therefore that these symptoms were due to an overstability of the oxy-haemoglobin resulting from acapnoea. A similar condition may be produced by excessive removal of carbon dioxide through forced breathing. The urine during this period of hyperpnoea differed conspicuously from that when the temperature was steady. The diuresis was not so excessive (probably modified in this way by the sweating). The urine, however, was alkaline and contained no ammonia but much bicarbonate and some ketone bodies. The decrease in the amount of urinary acid and ammonia was in proportion to the degree of the hyperpnoea. The loss of acid could not have occurred through the sweat as this was found to be alkaline with a pH of 7.9.

When the body temperature remained steady at 38.6°C. the hyperpnoea decreased to 12 litres per minute, the respiratory quotient to 0.81 and the alveolar carbon dioxide increased to 30.0 mm. Provided the temperature was not increased, no further respiratory disturbance occurred. There was a decrease in the urinary excretion of acid and ammonia, but they did not disappear as was the case during the period

†As there was little difference (0.1° C.) between the mouth and rectal temperatures the former were recorded.

of rapidly increasing body temperature with hyperpnoea.

Sweating began when the temperature rose above 37.2°. There was a progressive increase of the haemoglobin (up to a rise of 16%) in proportion to the amount of sweating, indicating an increased concentration of the blood. The pulse and pulse pressure rose with the body temperature but there were no general circulatory symptoms unless a considerable amount of weight was lost (2 kilos), when the pulse became small and rapid, with restlessness and other symptoms of circulatory failure. The circulatory responses showed individual variations.

The out-standing result obtained by Bazett and Haldane was the pronounced effect which a rapid increase in the body temperature had upon the respiration, the great hyperpnoea with lowering of the alveolar carbon dioxide pressure and (as will be pointed out later) consequently a decrease in the carbon dioxide content of the arterial blood. These phenomena would strongly suggest a pronounced sensitiveness of the respiratory centre to carbon dioxide when the body temperature, and therefore that of the brain, is elevated. This is supported by the experiments of Goldstein (1872). By enclosing the carotids in small metal water-jackets he was able, without changing the rectal temperature of the animal, to increase the temperature of blood going to the brain. He found when he did this that he raised the excitability of the bulbar centres, particularly of the respiratory centres. This was so pronounced that he was unable to produce apnoea by means of forced artificial respiration.

The effect of increasing the body temperature on the carbon dioxide pressure of the alveolar air was first investigated by Haggard.[3] He found, with an increase of body temperature produced by hot baths, that it was lowered and also that the amount in solution in the blood was lowered. He found no proportionate decrease in the alkali reserve. He therefore concluded that unless there was "some alteration in the dissociation constants, the pH was presumably lowered." Experiments *in vitro*, however, show at different temperatures no change in the carbon dioxide dissociation curve of the blood which was not accounted for by the alteration of the solubility of carbon dioxide and the concentration of H_2CO_3. Pemberton, Hendrix and Crouter[4] when studying the effects of "electric bakes," confirmed the fall in the alveolar carbon dioxide found by previous workers, while Pemberton and Crouter[5] in similar studies confirmed the increasing alkalinity of the urine and decreasing acidity of the sweat both in normal and arthritic persons.

Cajori, Crouter and Pemberton still further contributed to our knowledge of the effect of heat upon an acid-base balance. They were able on elevating the body temperature to demonstrate that the blood became more alkaline as represented by the pH and that the "alkali reserve" was increased as revealed by carbon dioxide dissociation curves. Consequent upon these disturbances compensatory changes occurred in the urine and sweat.

About the same time Koehler[7] made an extensive study of the acid-base equilibrium of the blood as affected by external heat. He studied and compared the effects of voluntary hyperpnoea, thermal fever and infective fever. He confirmed the results of Hill and Flack[8], Haggard[3], and Bazett and Haldane[2]. He also made an extensive study of the pH of the blood by electrometric methods. He found in acute infective fevers that the pH was uniformly higher during the febrile than in the afebrile period. There appeared to be a direct ratio between the degree of fever and that of the pH. On immersion in hot water baths, whereby the body temperature of the subject was raised to between 39.5° and 40.7°C. he confirmed the results of previous workers in regard to the hyperpnoea, increased pulse rate, decrease of alveolar carbon dioxide pressure, decrease of total carbon dioxide in the blood and the increase of the pH. In fact he increased the body temperature so rapidly as to induce tetany such as may result from voluntary hyperpnoea. He demonstrated that these symptoms could be prevented or removed by the inhalation of carbon dioxide. A previous demonstration of this by Bazett and Haldane[2] has already been mentioned.

The tetany which occurs after violent and sudden hyperpnoea would appear to be due to an acute alkalosis produced by the rapid removal of carbon dioxide. The basic ions are therefore left in the blood and tissues being more slowly eliminated by the urine, sweat, saliva and other secretions. To what an extent the sweat may eliminate carbon dioxide is not exactly known. Shierbeck determined that at a temperature between 29° and 33°C. 8.4 gms. per 24 hours were removed by this means. Above this point it rapidly increased—at 34°C. it was increased to 17 gms. per day and at 38.5°C. might amount to as much as 30 gms. As to whether this is a temporary or a permanent rate of elimination is not known, nor has the relative influence of steady and rapidly increasing febrile conditions been investigated. It may be that this is but a passing increase occurring during the period of rising temperature.

In this connection there have recently been reported some very interesting observations by Argyll Campbell[15]. He found that when he injected air under the skin and then immersed the animal in hot water that the partial pressure of carbon dioxide increased and when the animal was cooled it decreased. The significance of these phenomena is not at present clear.

So far we have been concerned chiefly with the effect of changes of body temperature upon the carbon dioxide, pH and allied conditions of the blood. It is important, however, to inquire in what manner if at all the combination of oxy-haemoglobin may be disturbed. *In vitro* experiments have indicated that as the carbon dioxide pressure decreases the oxy-haemoglobin dissociation curve moves to the left[9]. In other words, the less the partial pressure of carbon dioxide the greater the saturation of oxy-haemoglobin and the more tenaciously will it be retained at a given pressure of oxygen. On the other hand, it has been shown that there is also a shift in the oxy-haemoglobin curve with changes of temperature. If the temperature be increased the curve shifts in an opposite direction or to the right. This would indicate that as the temperature is increased so the saturation of oxy-haemoglobin is decreased at any particular pressure of oxygen, likewise as the temperature increases so the dissociation of oxygen from haemoglobin would occur more rapidly. In the present circumstances of lowered carbon dioxide content and pressure and increased temperature, we have apparently two conditions operating in opposite directions which might be expected to more or less neutralize each other. That this is the case in pathological conditions is not at all clear. During the past year observations have been carried out to investigate this point.‡ In the meantime, Koehlar[7] has suggested the possibility of loss of carbon dioxide being a factor in decreasing the oxygenation of the arterial blood under pathological conditions. He briefly discusses the theoretical reasons for such a phenomenon and reports two cases of cyanosis with pronounced alkalosis. The indirect evidence of improvement of symptoms with acid therapy is very suggestive, but it is unfortunate that he did not obtain data regarding the oxygen saturation of the arterial blood or the character of the oxy-haemoglobin curves in these cases. Morris[15] gives details of experiments in which the injection of sodium bicarbonate produced a lowering of the arterial oxygen saturation and an increase of the venous saturation, indicating not only an arterial anoxaemia but also a diminished dissociation of oxygen to the tissues.

The investigations which have been carried on in this department as to the oxy-haemoglobin curve in febrile conditions indicate a conspicuous difference between *in vitro* and *in vivo* experiments. They have confirmed the previous observations that there is an increased alkali reserve in infective fevers, in that both the pH and the carbon dioxide dissociation curve are distinctly shifted to the side of alkalosis, while the oxy-haemoglobin curves of blood taken during the febrile period have shown a distinct shift in the opposite direction.

With a different object in view, Uyeno[10] carried out experiments on cats immersed in hot water. The usual result of extreme hyperpnoea (with rapid and shallow respirations and also an increased oxygen consumption) was obtained, and in addition there was found to be a constant reduction in the arterial oxygen saturation. The hyperpnoea would appear to be readily accounted for. The reduction of arterial oxygen saturation might be explained either by the character of the breathing as suggested some years ago by Haldane, Meakins and Priestley[11] or possibly by a change in

‡These observations are presently to be published by Dr. A. C. White.

the chemical equilibrium of the blood as already mentioned by Koehler. An experimental investigation of this point would probably be of great clinical importance.

The experiments of Uyeno were undertaken to determine the effect of increased body temperature upon the minute circulation rate. He found that the volume of blood circulated per minute was increased by 20-30 per cent. Whether this increase in the circulatory minute volume was the result of a generally increased metabolism or the increased action of the respiratory pump was not determined. He came to the conclusion, however, that the effects of the warm water on the skin or the anoxaemia could not presumably explain it. He made observations on the character of the blood in the saphenous vein when the body temperature was raised by immersion in hot water. He found that the oxygen saturation of the blood in the vein was increased, while that of the blood in the right auricle was decreased. On the other hand, although the circulation rate through the saphenous area was definitely greater than normal it was not as proportionately accelerated as that of the general blood flow.

These results of Uyeno corroborate the findings of Meakins and Davies, that the oxygen saturation of the venous blood varies considerably, depending upon the temperature of the part from which the blood comes. Blood from the median basilic vein of the arm placed in hot water at 45° C. was found by them to be 94% saturated with oxygen, while when the arm was exposed to a temperature of approximately 15° C. the saturation was reduced to about 5%. These results were substantiated by Barcroft and Nagahashi[13], and by Pemberton, Hendrix and Crouter in "Electric-bakes."[4] Goldschmidt and Light[14] carried out similar experiments in more detail. They not only investigated the oxygen saturation of the haemoglobin from the deep veins at the elbow, but that from the superficial vessels at the back of the hand. They immersed the arm in water varying from 6° to 45° C. At the lowest temperature the percentage saturation of the haemoglobin closely approximated that of the arterial blood. This held not only for the blood obtained from the superficial veins but also from the deep veins presumably draining the deeper areas. With intermediate temperatures, however, the venous oxygen saturation was much decreased. On further increasing the temperature the oxygen saturation increased in proportion, gradually approximating the saturation of the arterial blood. We have found that the carbon dioxide content of the venous blood follows closely that of the oxygen saturation but in an inverse direction. In those instances where the oxygen saturation was increased so the carbon dioxide content was decreased, while on the contrary when the oxygen saturation was decreased the carbon dioxide content was increased.

These results might be explained upon either of two hypotheses—an increased blood-flow, or a decreased metabolism.

The experiments of Uyeno[10], and the other observations already reported, seem to prove conclusively that there is a greatly increased metabolism when the body temperature is raised by immersion in hot water. Therefore the increased saturation of the oxy-haemoglobin in the veins when a part is exposed to such conditions appears to be most probably due to a greatly increased circulation. It remains to account for this latter condition. The question arises as to whether the local increased blood flow results from the hyperaemic condition of the skin or whether it be due to the increased metabolism? The experiments of Uyeno would appear to give an answer to this question. On analysing his experiments 16 and 17 it is found in experiment 16 that the oxygen consumption in the saphenous area increased about 2.7 times while the blood flow increased about 6 times. In experiment 17 while the oxygen consumption remained about constant, the blood flow increased 3.3 times. It may be concluded therefore that when the temperature of a local area is increased by immersion in hot water, the increase in local circulation is in great part a direct result of the heat in producing a local hyperaemia.

Barcroft and Marshall[14] carried out some experiments on man to determine if the general circulation rate was influenced by the surrounding temperature. On exposure to cold (-1° C.) almost to the production of obvious shivering, they found that there was a regular increase in the circulation rate per minute which was very closely parallel to the increase of metabolism. (This was probably closely connected with the muscular movements resulting from the shivering). When the surrounding temperature was increased to about 40° C. the circulation rate increased, but to a much less extent and there was no appreciable change in the metabolism. The increased blood flow could quite readily be accounted for by the increased circulation through the skin. This in some of the experiments must have been considerable. As for instance on Marshall, the general blood flow increased from 3 to 6.8 litres per minute (127%) while the oxygen saturation of the mixed venous blood increased from 37 to 70 per cent. As there was practically no increase in the metabolism this increased blood flow was most probably in the superficial vessels and therefore by decreased utilization of oxygen in these areas the oxygen saturation of the mixed venous blood was raised by probably more than 100 per cent. (as when the surface of the body is exposed to room temperature the oxygen saturation of the venous blood from the superficial areas is probably much below that from the viscera).

Reference has been made to the findings of Bazett and Haldane that where there was extreme sweating the haemoglobin percentage increased from 10 to 16 per cent., indicating a proportionate concentration of the circulating blood. If this were always the case it would indicate a decrease in the total blood volume circulating at the moment. The effect of temperature upon the apparent blood volume is one of great interest. It was observed by the Royal Society Expedition to Peru[16] that the blood volume, as calculated by the carbon monoxide method, underwent considerable changes with the temperature of the atmosphere in which the subject was living; being greater in tropical than in more temperate climates. These observations were subsequently confirmed by experiments in a chamber where the air was artificially heated. In neither case was there any obvious change in the concentration of the blood as indicated by the haemoglobin percentage, although the total circulating haemoglobin appeared to increase conspicuously and at such a rate as could not be accounted for by fresh formation of the pigment. If this were not the case could it be accounted for by the fact that there was a considerable number of red blood corpuscles in the body to which the carbon monoxide was not readily accessible, but which could be thrust into the circulation if circumstances demanded? This question has to some extent been answered by Barcroft and Barcroft[17]. They found that the red blood cells of the spleen acquire CO very slowly and that the CO saturation of haemoglobin in this organ lags far behind that of the general circulation. They demonstrated in rats that after breathing CO for an hour the saturation in the spleen was not equal to that in the circulating blood. Conversely they found that the blood in the spleen gave up its CO much more slowly than that in the general circulation. It would appear, therefore, that it depends upon the acuteness of the sweating and the character of the environment as to what effect temperature may have upon the concentration of the blood and the apparent blood volume. It might be found in such experiments as Bazett and Haldane carried out, that there would be not only an increased concentration of the haemoglobin but also an increase in the total amount in the general circulation.

In regard to the effect of temperature upon blood volume and concentration the work of Barbour and his associates must here be considered. He[19] found with dogs that immersion in a hot bath (40° C.) produced a dilution of the blood solids, while exposure to cold or an increased cooling power reversed the process. He also demonstrated a distinct change in the brain volume, the brain becoming larger and apparently more ischaemic on exposure to cold, while the reverse happened with heat. All these results occurred even when the blood temperature remained normal. In animals with the spinal cord or brain destroyed the blood changes did not occur in spite of great fluctuations in the body temperature[20]. He comes to the tentative conclusion that the concentration and dilution of the blood under changes of temperature is an essential factor in the maintenance of a constant body temperature[21]. Lozinsky[22] continued this investigation with reference to the effects of "dry" and "moist" heat. He concluded that dogs in part regulate the body temperature by blood dilution—in "moist" air between 27° and 30° C. and in "dry" air between 37° and 39° C. In "moist" air temperatures above 33° C. and in "dry" air temperatures above 42° C. they regulate the body temperature very imperfectly and marked blood concentration occurs.

The evidence at present available would indicate a difference between the heat control methods in dogs and man. The experiments all go to show that in man there is a concentration of the blood solids and haemoglobin[23] on exposure to heat. This is most pronounced when there is a high humidity, under which conditions the cooling power of sweating is least efficient and therefore the loss of water through the skin is most abundant. Hunt[24], however, demonstrated that during exposure to "dry" heat there may be a conspicuous loss of weight (2.5 kilo) without evidence of an altered fluid content of the blood. It would appear that additional investigations are necessary under different conditions in man before this point can be settled.

The response to more prolonged and extreme changes of temperature particularly in association with muscular work does not particularly concern us at the moment. The consideration of these problems is more intimately connected with "water poisoning."

SUMMARY

The effects of increase or decrease of body temperature may be summarized as follows:

Respiratory Exchange and Metabolism.—If the body temperature be rapidly increased there is a pronounced hyperpnoea with rapid elimination of carbon dioxide, as a result of which the respiratory quotient may be above unity. If the body temperature be raised by the application of external heat or through infective processes there is an increase of metabolism in proportion to the increase of body temperature. If, however, the external heat be not sufficient to raise the body temperature the respiration and metabolism remain practically constant. If the increase of body temperature be too severe and prolonged the metabolic processes will gradually become less than normal and eventually may cease. The application of cold in such a manner as to increase the cooling power of the body produces a very pronounced increase of metabolism which will continue until the thermogenic power becomes exhausted and then a decrease of metabolism will take place.

Circulation.—On exposure to heat there is a tachycardia, lowering of the diastolic blood pressure, an inconstant increase of the systolic pressure and an absolute increase of the pulse pressure. Coincident with these changes there is an increase in the total blood flow which is proportional to the increase in the cutaneous circulation. If, however, the body temperature be raised the increased blood flow is greater than can be so accounted for, and is probably the result of the increased metabolism. When the dilatation of the cutaneous capillaries becomes extreme there develops deficiency of the return flow of blood to the heart with signs of circulatory failure. When the body is exposed to cold there is an increase of the circulation which appears to be directly parallel to the increase in metabolism.

Blood.—The removal of carbon dioxide resulting from the hyperpnoea leads to a pronounced decrease in the partial pressure and to a smaller decrease in the carbon dioxide content of the arterial blood. This usually leads to an uncompensating alkalosis and an increase in the pH of the blood. The effect on the oxyhaemoglobin is more indefinite but none the less important. Some direct and indirect observations suggest a decrease in the combining power while others might indicate an instability in regard to the tissue. The common result, however, would point towards a more or less pronounced decrease in utilization of oxygen by the tissues. There also results a concentration of the blood resulting from the loss of fluid either through diuresis or sweating. This is demonstrated by the increase of the haemoglobin percentage and the concentration of the blood solids. This concentration of the blood is more pronounced with "moist" than with "dry" heat.

Urine.—On immersion in hot water there is produced a copious diuresis. This is greatest when the body temperature remains normal and is probably caused by the pressure of the water. If the body temperature be raised and profuse sweating occurs the diuresis is not so pronounced. With the rise of body temperature and the hyperpnoea, the urine changes from an acid to an alkaline reaction, the amount of acid and ammonia eliminated may be reduced to zero and the urine may contain bicarbonates. This change towards the alkaline

reaction with suppression of acid and ammonia excretion is in proportion to the hyperpnoea.

Sweat.—When the temperature of the hot bath is above 35° C. sweating begins. If the body temperature be raised the pH of this secretion becomes more alkaline and it contains an increased quantity of carbon dioxide.

The ideal in therapeutics is to know in the first instance in what manner the human organism is disturbed by either functional or structural changes, and the etiological factor producing such altered conditions. Usually, if the cause be removed, the organism will automatically and rapidly recover. On the other hand, it is often necessary to adopt such measures as will aid the organization to rectify processes which, if carried beyond a certain point, may menace its well being.

Under all these conditions it should be our endeavour to accurately determine the manner of the disturbances and the method of operation of the remedies employed; the twilight of empiricism being replaced by the sunlight of critical knowledge based upon accurate observations.

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A Simple Method of Prescribing Diabetic Diets.—George Baehr, Herman Lande and Lulu G. Graves, New York, offer a series of twelve test diets devised for the general practitioner in medicine, to assist him to prescribe accurate diabetic diets without the use of mathematical formulas. With its aid, the physician should be able to prescribe well balanced diets of known food value and immediately write out the menus for three meals a day with the accuracy of a trained dietitian. In preparing this table the authors have modified the one of Joslin so as to make it conform to the present day needs of the high fat, low protein diets for patients with diabetes. They therefore preserve a proper antiketogenic balance. They contain a constant minimum amount of protein and a moderately large amount of fat. The carbohydrate foods are in one group and the protein and fat in another. This makes it possible to increase the carbohydrate foods in each succeeding test diet, whereas the quantities of protein and fat remain practically constant. The fat is reduced in the higher diets only in order to keep the total food values below the needs of the individual, and so maintain a moderate undernutrition during the test period. The twelve diets are called test diets, for they are designed to be used only during the first week or two, in order to eliminate the patient's glycosuria, reduce his blood sugar to a more normal level, and then test his maximum glucose burning ability. After this has been accomplished, the diet is increased in accordance with certain rules, this final, more adequate diet being called the permanent maintenance diet. —*Jour. Am. Med. Assoc.*, May 10, 1924.

Papillary Epithelioma of the Kidney Pelvis.—The chief symptom in the case reported by Carl Eggers and Joseph Felsen, New York, was hematuria, and, as the result of loss of blood, weakness and dyspnea. A tumor was not palpable, owing to the fact that the kidney was not enlarged. The roentgen-ray findings were negative. The cystoscopic findings showing the blood to come from the left side, together with the marked diminution of the kidney function on the affected side, were of great value. The kidney was removed. Four years after operation, there had been no return of symptoms.—*Jour. Am. Med. Assoc.*, May 3, 1924.